



NOTES

SUPRAVENTRICULAR TACHYCARDIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Abnormally fast heart rhythms due to inappropriate electrical activity in upper portion of heart, atria/atrioventricular (AV) node
- Ventricles contract > 100 beats per minute, pathology originates above ventricles
 - Ventricles protected by gating at AV node

CAUSES

- Reentry conductive loops
- Increased automaticity
- Triggered activity

RISK FACTORS

- **Cardiac:** coronary artery disease, heart failure
- **Non-cardiac:** chronic obstructive pulmonary disease (COPD), pulmonary embolism, alcohol abuse, hyperthyroidism

SIGNS & SYMPTOMS

- Palpitations, chest pain, anxiety, dyspnea, syncope, lightheadedness

DIAGNOSIS

LAB RESULTS

- Electrolytes, thyroid stimulating hormone levels

OTHER DIAGNOSTICS

- ECG

TREATMENT

- See individual disorders

MEDICATIONS

- Calcium channel blockers, beta blockers, anticoagulants

SURGERY

- Catheter ablation

VISIT...

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ATRIAL FIBRILLATION

osms.it/atrial-fibrillation

PATHOLOGY & CAUSES

- **Rapid, irregular** (no discernible rhythm) **heart rate**
- Progression
 - **Paroxysmal**: intermittent rhythm, may revert back to sinus rhythm spontaneously
 - **Persistent**: > seven days, requires intervention to convert back to sinus rhythm
 - **Permanent**: long-standing atrial fibrillation, cardioversion unsuccessful

CAUSES

- Disorganized waves of atrial depolarization, exact mechanisms not well understood
 - Regular impulses of sinus node overwhelmed by rapid electrical discharges from various sources (automatic foci, multiple reentry phenomena)
 - Arise from left more than right atrium

RISK FACTORS

- **Old age**: affects 4% 60–70, 14% > 80
- Obesity, diabetes mellitus, excessive alcohol consumption, genetic predisposition
- **Cardiovascular disease**: **heart failure**, hypertension, coronary artery disease, non-rheumatic mitral regurgitation, mitral valve prolapse, rheumatic heart disease, damaged atrial myocytes
- Increased catecholamine levels
- Lung disease
- Hyperthyroidism

COMPLICATIONS

- **Thromboembolic events**, heart failure, hypotensive shock

SIGNS & SYMPTOMS

- May be asymptomatic
- Dyspnea, fatigue, palpitations, lightheadedness, weakness, chest pain, hemodynamic shock

DIAGNOSIS

LAB RESULTS

- **Thyroid stimulating hormone (TSH)** levels: exclude hyperthyroidism

DIAGNOSTIC IMAGING

Transthoracic echocardiogram

- Evaluate atrial, ventricular size; valvular disease; left ventricular function; pericardial disease

Transesophageal echocardiogram

- Evaluate for atrial thrombi

OTHER DIAGNOSTICS

ECG

- **Absent P waves**
- **Irregularly timed QRS complexes** (irregular R-R intervals)
- **No sawtooth wave** in atrial fibrillation

TREATMENT

MEDICATIONS

Anticoagulation

- E.g. warfarin, dabigatran, apixaban, rivaroxaban
- **CHA₂DS₂-VASc/CHADS₂ score**
 - Estimate risk of stroke in non-rheumatic atrial fibrillation; higher score = greater risk of stroke
 - **Score 0 (biological male)/1 (biological female):** low risk, no anticoagulation recommended
 - **Score 1 (biological male):** moderate risk, consider anticoagulation
 - **Score ≥ 2:** high risk, anticoagulation recommended
 - See table of scores

Rate control

- < 100 beats per minute
- **Beta blockers** (preferably β₁ selective)
- **Non-dihydropyridine calcium channel blockers** (e.g. diltiazem, verapamil)
- **Digoxin**

Chemical cardioversion

- Administer antiarrhythmic medication
- Class Ic antiarrhythmics
- Class III antiarrhythmics
- Maintenance of sinus rhythm after cardioversion
 - Class Ic antiarrhythmics
 - Class III antiarrhythmics

OTHER INTERVENTIONS

Rhythm control

- Restore sinus rhythm via **cardioversion**

Electrical cardioversion

- Defibrillator for synchronization

Catheter ablation

- Destruction of heart regions responsible for abnormal impulses

CHA₂DS₂-VASc SCORING SYSTEM

	CONDITION	POINTS
C	Congestive heart failure: left ventricular systolic dysfunction	+1
H	Hypertension: consistently > 140/90 mmHg	+1
A₂	Age: ≥ 75 years	+2
D	Diabetes mellitus	+1
S₂	Prior stroke, TIA, thromboembolism	+2
V	Vascular disease: peripheral artery disease, myocardial infarction, aortic plaque	+1
A	Age: 65–74 years	+1
Sc	Sex: biological female	+1

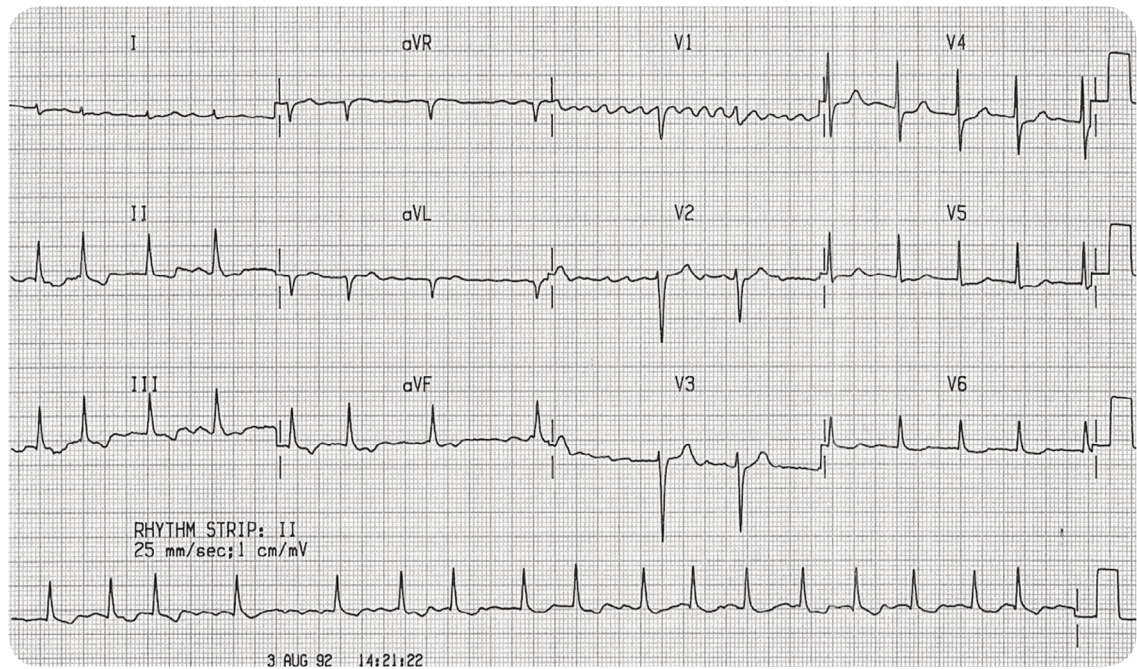


Figure 20.1 An ECG demonstrating atrial fibrillation.

ATRIAL FLUTTER

osms.it/atrial-flutter

PATHOLOGY & CAUSES

- Atria depolarize regularly at **very high rates** (200–350bpm), appear to flutter

- Isthmus-independent
- Reentrant circuit develops in either atrium
- Associated with variety of reentry loops (common after incomplete atrial ablation procedures, right atrial surgical scars)

TYPES

Typical atrial flutter (AKA Type 1 flutter)

- More common
- **Single reentrant circuit, right atrium**
- **Isthmus-dependent:** reentry circuit crosses cavotricuspid isthmus
- Circles tricuspid annulus (ring), usually counterclockwise (viewed from below)
- Cavotricuspid isthmus tissue propagates signal slower than surrounding tissue → circuit loops → slows propagation → surrounding tissue exits refractory period

Atypical atrial flutter (AKA Type 2 flutter)

- Less common

CAUSES

- **Reentrant electrical signal** from **either atrium**
- Reentrant signal loops back on itself → overrides normal sinus rhythm → establishes **endless loop** of stimulation
- Underlying disease (e.g. heart failure, valvular disease, hypertension, pulmonary disease) → heart cells less electrically stable → **alters refractory periods** → increased risk of reentrant circuits
- Reentrant circuits initiated by premature atrial contraction (PAC) → partial premature contraction, normal tissue relaxes → wave of stimulation propagates → normal tissue

contracts, premature tissue recovers → chance of reentrant circuit, stimulation wave doubles back on itself

RISK FACTORS

- Diseases that change atrial heart cell properties → differing electrophysiological properties in adjacent areas → reentry circuit
- Ischemia, fibrosis, previous myocardial infarction, heart failure, high blood pressure, diabetes, valvular heart disease, obstructive sleep apnea

COMPLICATIONS

- **Heart failure**, thromboembolic events, atrial fibrillation

SIGNS & SYMPTOMS

- Palpitations, tachycardia, fatigue
- Pain/tightness/discomfort in chest
- Heart failure
 - Exercise intolerance
 - Difficulty breathing at night/while lying flat
 - Edema of legs, abdomen

DIAGNOSIS

DIAGNOSTIC IMAGING

Echocardiogram

- Evaluate size of right, left atria, ventricles
- Detect pericardial/valvular heart disease
- Decreased ejection fraction (% of blood pumped by heart per contraction)

LAB RESULTS

- Serum electrolytes
- Renal function
- *Thyroid stimulating hormone (TSH) levels:* exclude hyperthyroidism

OTHER INTERVENTIONS

ECG

- Typical **P waves** absent
- **Typical atrial flutter:** **P waves**, **saw tooth shape** (F waves) localised to leads II, III, aVF
- **Atypical atrial flutter:** atrial activity (sawtooth waves/otherwise) may occur **anywhere**, dependent on reentrant circuit location
- Ventricular rate usually 1/2 atrial flutter rate (even ratios 2:1, 4:1 more common than odd, 3:1, 5:1)
- **1:1 atrial:** catecholamine excess, presence of accessory bypass tract/class 1A, 1C antiarrhythmic drug therapy

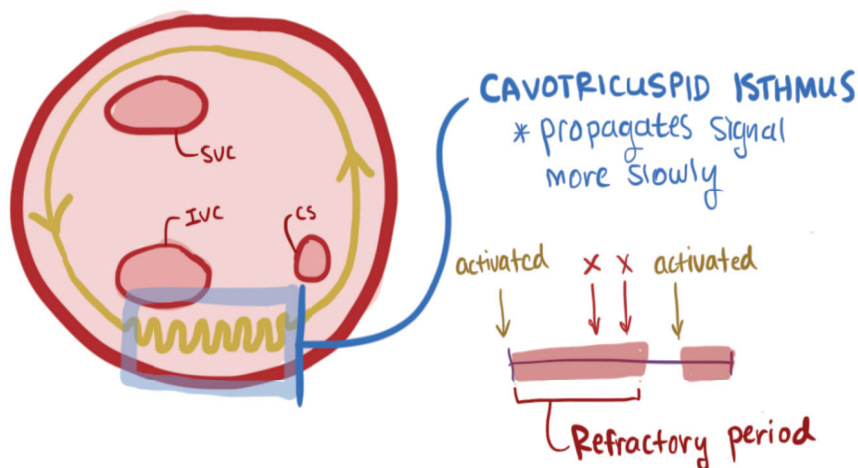


Figure 20.2 An electrocardiogram demonstrating atrial flutter with a 3:1 AV nodal block. The atrial trace demonstrates a characteristic sawtooth pattern.

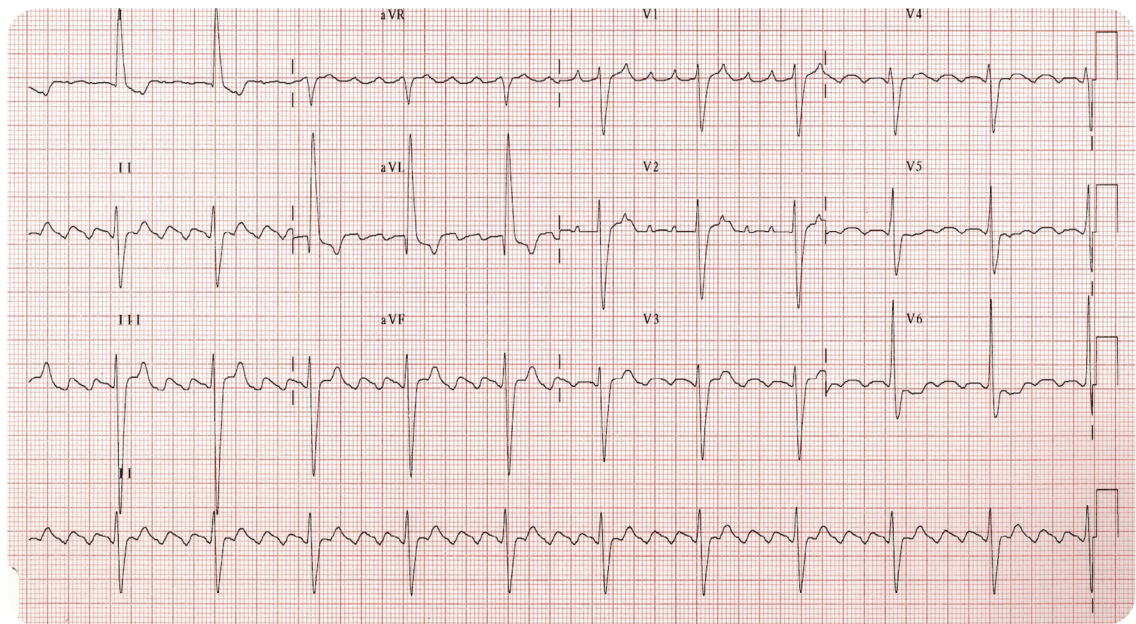


Figure 20.3 An electrocardiogram demonstrating atrial flutter with a 3:1 AV nodal block. The atrial trace demonstrates a characteristic sawtooth pattern.

TREATMENT

MEDICATIONS

- **Anticoagulants** (reduce chance of clot formation), beta blockers/calcium channel blockers (control rates of ventricles)

SURGERY

Radiofrequency catheter ablation

- Cavotricuspid isthmus no longer able to carry electrical signal, **prevents reentry**

OTHER INTERVENTIONS

Electrical cardioversion

- Depolarize atrial tissue, **resynchronize contraction**

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA (AVNRT)

osms.it/av-nodal-reentrant-tachycardia

PATHOLOGY & CAUSES

- Heart rate disturbance due to **accessory pathway** in/near AV node
- Electric conduction splits into **two pathways** → forms loop
 - Alpha pathway**: slow conduction, short refractory period
 - Beta pathway**: fast conduction, long refractory period

TYPES

Slow-fast/"typical" AVNRT

- Anterograde conduction to ventricles via slow pathway (alpha)
- Retrograde to atria conduction via fast pathway (beta)
- Depolarization down both pathways → reaches end of beta pathway first → **signal splits**
 - Travels to ventricles** → contraction
 - Travels up alpha pathway** → meets slow signal → signals cancel each other out
- Depolarization wave from premature beat reaches AV node → refractory fast (beta) pathway → signal initially down alpha pathway only → **splits**
 - Travels to ventricles** → contraction
 - Travels up beta pathway** → signal travels down alpha pathway, beta pathway comes out of refractory period → signal reaches end of alpha pathway → splits back up beta pathway

Fast-slow/"atypical" AVNRT

- Anterograde conduction via fast pathway, retrograde conduction via slow pathway

RISK FACTORS

- Biologically-female individuals → 75% of cases, emotional stress → **alcohol use disorder**, hyperthyroidism, electrolyte disturbances

SIGNS & SYMPTOMS

- Palpitations**, transient chest pain, bouts of transient tachycardia, transient hypotension, (pre)syncope

DIAGNOSIS

LAB RESULTS

- Thyroid function
- Serum electrolytes

OTHER DIAGNOSTICS

ECG

- Tachycardia 140–280bpm
- Absent P waves**
- P waves **immediately before/after QRS complex**
- P waves inverted/retrograde**
- R' waves** (small secondary R waves)

TREATMENT

MEDICATIONS

- Adenosine**, **beta blockers**, calcium channel blockers to slow AV node conduction

OTHER INTERVENTIONS

Radiofrequency catheter ablation

- Definitive treatment
- Ablation of slow alpha pathway

Slow AV node conduction

- Vagal maneuver (carotid sinus massage/ Valsalva maneuver) → activates vagus nerve

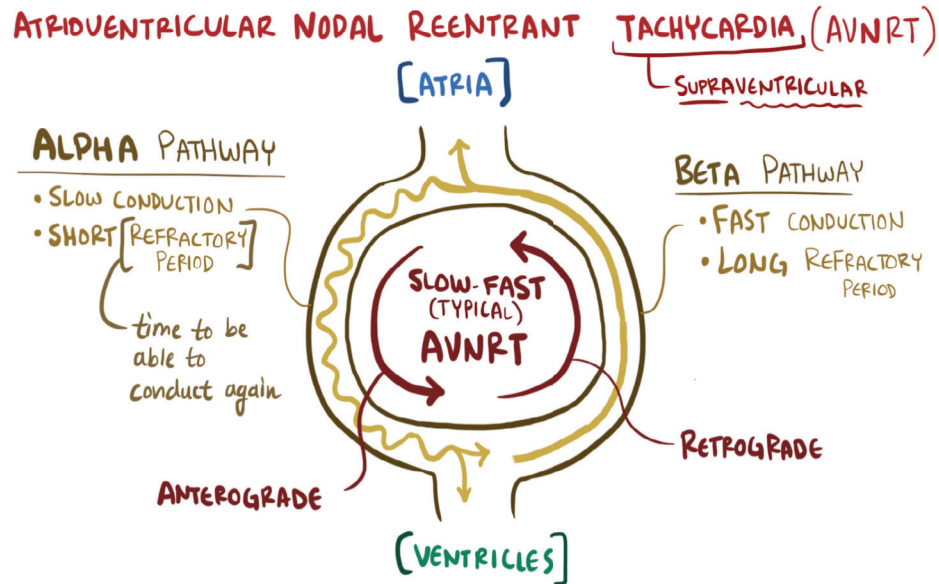


Figure 20.4 Illustration depicting path of electrical conduction in AV node during slow-fast AVNRT.

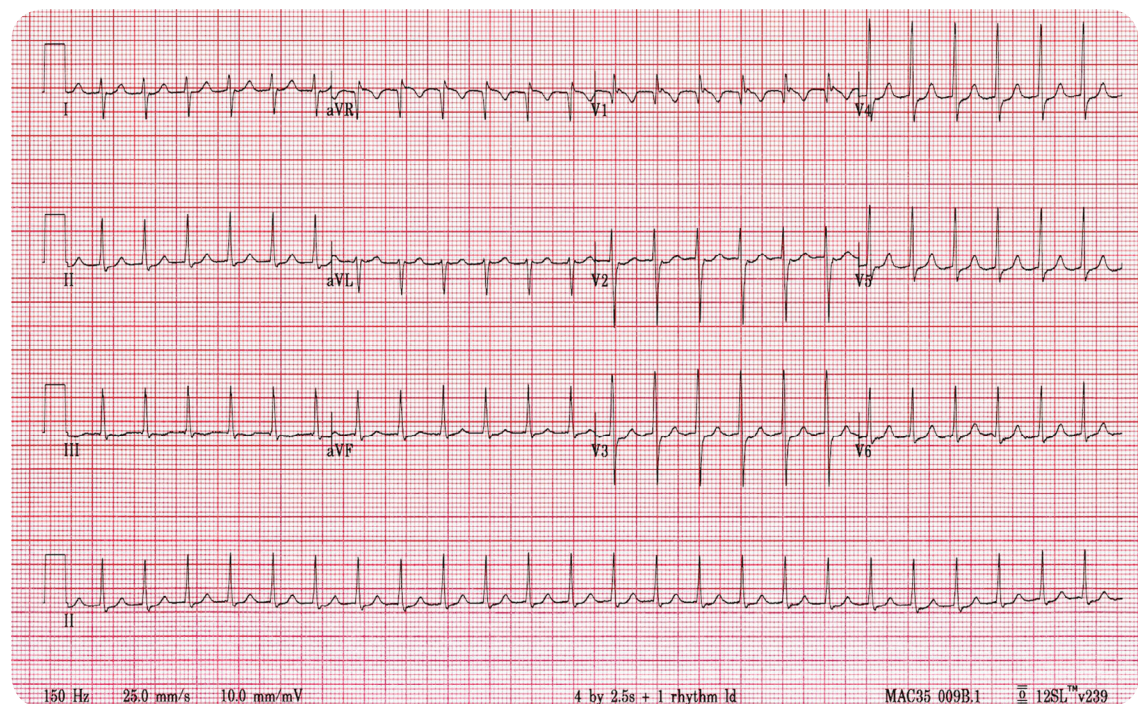


Figure 20.5 An ECG demonstrating typical (slow-fast) AVNRT. R waves are best seen in lead V1.